transcriptase. "EM 1," as used herein, refers to a deletion mutant of endostatin, wherein the last nine amino acid residues have been deleted (*i.e.*, NSFMTSFSK (SEQ ID NO: 25)), and the term is intended to include fragments, mutants, homologs, analogs, and allelic variants of the amino acid sequence of SEQ ID NO:2). Although EM 1 was originally cloned from mouse nucleic acid, it performs better than intact type endostatin (*i.e.*, endostatin that has not been mutated) in standard assays. The term EM 1 is therefore intended to include any mammalian sequence substantially similar to EM 1 as described herein, as well as mammalian EM 1 fragments, mutants, homologs, analogs and allelic variants of the mammalian EM 1 amino acid sequence. Also, specifically encompassed by the present invention are human endostatin mutants, and more specifcally, the human deletion mutant equivalent of EM 1.

Please replace the paragraph at page 71, line 3 with the following paragraph:

EM 1, a novel anti-angiogenic protein, and a deletion mutant of endostatin, is described, as well as methods of making EM1, therapeutic compositions comprising EM1, and methods for using those compositions.

Amendments to the specification are indicated in the attached "Marked Up Version of Amendments" (pages i -ii).

In the Claims

Please cancel Claim 1.

Please amend Claims 3, 4 and 35.

3. (Amended) The isolated EM1 of Claim 2, wherein the C-terminus of the isolated EM 1 comprises SEQ ID NO: 24.

4. (Amended) The isolated EM 1 of Claim 2, wherein the deletion of nine consecutive amino acids comprises SEQ ID NO: 25.